# EXHIBIT A

#### EXPERT DISCLOSURE OF WILLIAM BANNER, JR., M.D., PH.D.

Re: Glenn Burton, Jr.

#### I. Professional Qualifications

I am a physician and toxicologist, Board Certified in Medical Toxicology, Pediatrics and Critical Care Pediatrics. Currently, I am an attending physician in critical care pediatrics for Integris Baptist Medical Center and am the Medical Director of the Oklahoma Center for Poison and Drug Information in Oklahoma City. I have served on the Board of Directors of the American Association of Poison Control Centers since 2010 and am currently the President of that Board. Prior to moving to Oklahoma City, I was Medical Director of the Children's Hospital at St. Francis Hospital in Tulsa, Oklahoma, Co-Director of the Saint Francis Pediatric Intensive Care Unit, and also Medical Director of the Oklahoma Poison Control Center. I am a Past Chairman of the American Board of Medical Toxicology; Past President of the American Academy of Clinical Toxicology; and a Fellow of the American Academy of Pediatrics, the American College of Critical Care Medicine, the American College of Medical Toxicology, and the American Academy of Clinical Toxicology. I was the recipient of The ACMT Matthew J. Ellenhorn Award September 25, 2011, Washington, DC and The AACT Lifetime Career Achievement Award October 20, 2014, New Orleans, LA.

As a toxicologist and critical care pediatrician, I care for and treat children with a variety of serious medical issues, including elevated levels of potentially toxic substances. Through my education and 30+ years of clinical practice, I am familiar with the accepted methodology for differential diagnosis and etiology; child health and development; the scientific, medical, and toxicological literature on lead and other neurotoxins, including the risks associated with elevated lead levels in children, lead's potential adverse health effects and diagnosis of lead toxicity, pathways of lead intake, lead absorption, biokinetics, sources of lead in the environment, and other toxins and environmental factors affecting child health and development.

I have published more than fifty articles on the toxicity or pharmacology of various substances, including metals, and have studied lead chelation compounds. As a member of the American Academy of Pediatrics Committee on Drugs, I was the primary author of the 1995 treatment guidelines for children with elevated blood lead levels. I served as a member of the Centers for Disease Control and Prevention's ("CDC") Lead Poisoning Advisory Committee from 2002 to 2005. As a toxicologist and pediatrician, I am familiar with the scientific, medical, and toxicological literature on lead toxicity, including the risks associated with elevated lead levels in children with respect to health and cognitive, behavioral, and emotional development.

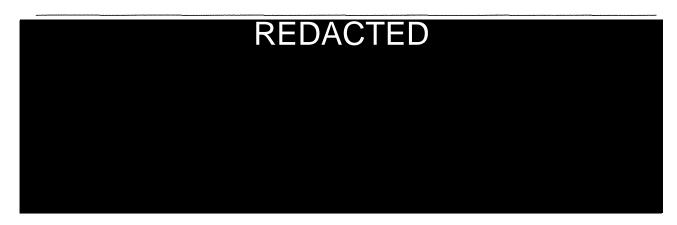
A copy of my curriculum vitae is attached as Tab A. A list of testimony and statement of compensation is attached as Tab B.



#### **OPINION THREE**

White lead carbonate pigments in paint in Glenn Burton's house did not present a uniform risk of harm to him.

The risk of lead toxicity depends not on the mere presence of a lead source, but rather on whether lead from any source actually gets into the body in a form that can be distributed to the potential targets of toxicity (bioavailability) at a sufficient concentration and for a sufficient



duration. The way a poison or toxin enters a person is called an exposure pathway. Pathways of exposure can be inhalation, ingestion, or topical absorption. The pathways for lead ingestion in children are inhalation of lead in air or dust, or ingestion of lead. For white lead carbonate as a pigment, this means that it must get from the paint on the wall (often covered by many other layers of paint) ingested by a child, and absorbed into the bloodstream.

As an initial matter, intact lead paint is not a hazard or pathway; 80 it does not present a risk to human health and is not considered to be a hazard in the laws and regulations promulgated by the U.S. Congress, the U.S. Environmental Protection Agency ("USEPA"), the U.S. Department of Housing and Urban Development, the State of Wisconsin, and the City of Milwaukee. This common sense notion is supported by the results of studies of children living in homes with high concentrations of lead-based paint yet who do not have elevated blood lead levels. Here, although Plaintiff's experts identify white lead carbonate in paint in the houses, there is no evidence that ingested white lead carbonate pigment: it is generally in the lowest layers of paint, there is no evidence that this paint was accessible to at the time he lived in the houses, and there is no evidence that it was a predominant source of lead in the dust in those houses. Plaintiff's experts also report many other layers of paint containing lead, but not identified as white lead carbonate. And as noted above, there were only two areas of deteriorated paint found by the MHD at the house, and there is no evidence that ingested lead from the few areas required to be repainted.<sup>81</sup> Based on the record, no pathway of exposure has been determined to a reasonable degree of medical certainty in this case, let alone a pathway tying any specific layer of paint containing white lead carbonate pigment to inhalation or ingestion by this child.

Beyond condition and accessibility of any white lead carbonate containing lead paint, not all lead sources pose equivalent risks of harm. The extent to which lead can get into the body, the blood, and other biological compartments varies depending upon the type, or species, of lead. Absorption, distribution and excretion of lead also depends upon basic physio-chemical properties, such as its condition, matrix, and particle size. For example, a metal object containing lead that sits in stomach acid creates more bioavailable lead than an equivalent object that passes through. Likewise, an object containing a high concentration of metallic lead that sits in stomach acid creates more bioavailable lead than a paint chip with a lower concentration of lead that passes through.

With respect to similar sources like lead pigments in paints, lead chromates are less bioavailable than lead carbonates; larger particle sizes are less bioavailable than smaller particles; and white lead carbonates in a matrix (such as paint) or as a component of intact lead-based paint have low potential bioavailability relative to pure white lead carbonates in oil or lead particles in dust or

<sup>&</sup>lt;sup>80</sup> 40 CFR Part 745, 66 Fed. Reg. 1206 (Jan. 5, 2001); Wis. Stat. § 254 *et seq.*, especially 254.11 (4h), (8g); Wisconsin H.F.S. Regulations § 163.42; Milwaukee Code Ordinances § 66-20 *et seq.*, especially §§ 66-21-15, 66-22-a, and 66-22-7 through 66-22-10.

<sup>81</sup> BurtonG-MHD-0000015-35.

<sup>&</sup>lt;sup>82</sup> See e.g., D. Barltrop et al., Absorption of Different Lead Compounds, POSTGRADUATE MED. J. 51:805-09 (1975); National Research Council, Committee on Measuring Lead in Critical Populations, Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations, 127, 140 (1993)

soil. <sup>83</sup> Similarly, lead dust from deteriorated paint would be more bioavailable after ingestion than lead from a paint chip, assuming the paint chip did not get caught up in the stomach for an excessive length of time. Thus, all lead sources, including white lead carbonate pigments and paints, do not present equivalent risks of harm due to differences in bioavailability.

Because of this, white lead carbonates present in a layer of paint, intact or otherwise, will not present a uniform risk to children in the home. The chemical properties of the paint in and around the lead will influence how much of the lead the body will absorb and how much it will excrete. How the body ingests the lead also matters. Toxins that are inhaled and retained in the lungs (such as airborne lead) are mostly absorbed. On the other hand, toxins that are ingested can be more readily expelled by the body. Similarly, the manner of deterioration or degradation leading to ingestion will impact the risk. If the paint particles containing lead are very small, less than 50 microns in size, they are more bioavailable than larger particles containing white lead carbonate. Thus, paint "chips" or "flakes" present less of a risk when compared to similar amounts of paint that has been sanded (or otherwise broken down into smaller particles). <sup>84</sup>

Plaintiff's expert, Dr. Mushak, states that all lead is the "same" once it is incorporated into the blood stream. This observation misses the point of toxicological risk. It is the process and efficiency of intake and absorption -- as opposed to excretion -- that dictates how much risk that a substance may present after it is ingested or inhaled. If the body excretes the entire amount of the toxin, there is no injury to the body. If only a small amount of the toxin is retained, then the risk is comparatively less. And, for white lead carbonate, there are many different facets to determine whether the body will absorb the lead or excrete it, including the matrix and size in which it is found.

In addition to bioavailability and the amount of bioavailable lead ingested, other factors impact lead's potential toxicity for a given individual, including: intake pattern (e.g., amount, frequency and duration); lead concentration; and characteristics of the individual potentially at risk for toxicity.

#### V. Conclusion

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<sup>&</sup>lt;sup>83</sup> See e.g., D. Barltrop et al., Absorption of Different Lead Compounds, POSTGRADUATE MED. J. 51:805-09 (1975); Michael V. Ruby et al., Lead Bioavailability: Dissolution Kinetics Under Simulated Gastric Conditions, ENVTL. SCI. TECHNOL., 26(6):1242-48 (1992); Stan W. Castee et al., Bioavailability of Lead to Juvenile Swine Dosed with Soil from the Smuggler Mountain NPL Site of Aspen, Colorado, FUNDAMENTAL AND APPLIED TOXICOLOGY, 36:177-87 (1997); EPA, Lead Identification of Dangerous Levels of Lead; Final Rule, 40 CFR Part 745, 66 Fed. Reg. 1206 (Jan. 5, 2001).

<sup>&</sup>lt;sup>84</sup> David E. Jacobs, U.S. Department of Housing and Urban Development, *Guidelines For The Evaluation And Control Of Lead-based Paint Hazards In Housing*, Appendix 11 (2004); see also John R. Kominsky et al., US EPA Project Summary, *Field Demonstration of Lead-Based Paint Removal and Inorganic Stabilization Technologies* (2002).

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William Banner, Jr., M.D., Ph.D.